What effect does consuming natural (ruminant) vs. synthetic (industrially hydrogenated) trans fatty acids have on LDL-, HDL- and Non-HDL cholesterol?

Conclusion

Limited evidence is available to support a substantial biological difference in the detrimental effects of industrial trans fatty acids (iTFA) and ruminant trans fatty acids (rTFA) on health when rTFA is consumed at seven to ten times the normal level of consumption.

Grade: Limited

Overall strength of the available supporting evidence: Strong; Moderate; Limited; Expert Opinion Only; Grade not assignable For additional information regarding how to interpret grades, click here.

Evidence Summary Overview

Three studies were reviewed to determine the effect of ruminant (rTFA) vs. industrially-produced trans fatty acids (iTFA) on low-density lipoprotein cholesterol, (LDL-C), high-density lipoprotein cholesterol (HDL-C) and nonHDL-C: One non-systematic reviews and two randomized controlled trials (RCTs).

It is well-documented that synthetic iTFA adversely effects LDL-C, HDL-C and non-HDL-C, but evidence is very limited that natural ocurring rTFA at levels typically consumed have any effect on cardiovascular disease (CVD) or coronary heart disease (CHD) risks. Based upon the results of two, small, well-designed crossover studies (Chardigny et al, 2008; Motard-Belanger et al, 2008), high intakes of rTFA (10.2g-12g per day) do not show consistent and different effects from synthesized iTFAs. One small RCT (Chardigny et al, 2008) found rTFA intake compared to iTFA intake increased both LDL-C and HDL-C in women, but not in men. This finding does not allow for a conclusion that there is any change in risk between iTFA and rTFA, since these lipid changes should not change theCVD risk appreciably.

Jakobsen et al, 2006 reviewed three prospective cohort studies, one case-control study and one descriptive study using CHD end-points and reported no significant (NS) difference in associations betweenrTFA and iTFA, corroborating the studies evaluating their effect on lipids and lipoproteins. Omen et al, 2001 confirmed these findings in a cohort of 667 Dutch men.

These data taken together, based upon very limited studies, indicate that there is insufficient evidence to suggest rTFA and iTFA be considered differentially in their metabolic effect. TotalTFA intake should be considered the target for dietary change.

Evidence Summary Paragraphs

Chardigny JM et al 2008, in a positive quality randomized, double-blind, controlled, cross-over trial that compared the effects of TFAs from industrially-produced and natural sources on HDL-C and LDL-C, lipoprotein particle size and distribution, apolipoproteins and other lipids in 40 (21 women, 19 men) healthy, normolipidemic subjects in France. The eight-week intervention consisted of two, three-week experimental periods and a one-week run-in and one-week wash-out period. The experimental diet incorporated either rTFA or iTFA (11-12g per day, representing approximately 5% of daily energy) consumed daily in form of 20g butter, 100g cheese and 22g cookies. Compliance was assessed via questionnaire and plasma assay for TFA in plasma cholesteryl-esters. Compared with TFAs from industrially produced sources, TFAs from natural sources significantly increased HDL-C (P<0.012) and LDL-C (P<0.001) in women, but not in men. In women, an increased concentration of large LDL-C particles was significant (P=0.009). Plasma concentrations of total cholesterol (TC) and triacylglycerol (TG) were also significant higher (<0.001 and P=0.001, respectively) in women consuming TFAs from natural vs. industrial sources. The TC to HDL-C ratio was NS increased.

Jakobsen et al, 2006, in a negative quality narrative reviewed the findings of five epidemiological studies that investigated the effects of different quintiles of intake ofrTFA and iTFA on CHD risk factors. Three prospective cohort studies, one case-control and one descriptive study were reviewed. Two of the three prospective cohort studies found an inverse association between energy-adjusted rTFA intake and risk of CHD. Willett WC et al, 1993, found that the relative risk (RR) of CHD for the highest vs. the lowest quintile of energy adjusted rTFA was 0.59 (95% CI 0.30-1.17) and Pietinen P et al, 1997, found that the relative risk of coronary death for the highest vs. the lowest quintile of energy adjusted rTFA was 0.83 (95% CI 0.62-1.11); and a case-control study (Ascherio A et al, 1994) found that theRR of myocardial infarction (MI) for the higher vs. lowest quintile of energy-adjusted rTFA intake was 1.02 (95% CI 0.43-2.41). Those findings might imply that intake of rTFA, as C18:1,t11 (vaccenic acid) is innocuous or even protective against CHD. One prospective cohort study (Oomen CM et al, 2001) found NS direct associations between intake of rTFA and iTFA and risk of CHD; i.e., for 0.5% higher level of energy intake from rTFA, the RR of CHD was 1.17 (95% CI 0.69-1.98) and, for iTFA, the RR was 1.05 (95% CI 0.94-1.17). Authors recommended that more controlled metabolic studies on the effect of intake of total and specific rTFA on CHD risk factors and more epidemiological studies of intake of rTFA and risk of CHD, assessing association for both absolute and energy-adjusted intake, be conducted. Method of selection of articles reviewed was not defined.

Motard-Bélanger A et al, 2008, in a positive quality double-blind, randomized, crossover controlled feeding trial that compared the effects of rTFA and iTFA on plasma LDL-C concentrations and other CVD risk factors. Thirty-eight male normolipidemic Canadian subjects (36 white and two black) were fed four experimental isoenergetic diets each lasting four weeks. The following diets were tested:

1. High rTFA (10.2g per 2,500kcal)
2. Moderate rTFA (4.2g per 2,500kcal), high iTFA (10.2g per 2,500 kcal) and low in TFA from any source (2.2g per 2,500kcal) (control diet).

Each diet was separated by a wash-out period of three to 12 weeks. All diets were identical in terms of menus, calories and macronutrient composition. Ruminant TFA andiTFA provided 3.6% of daily energy intake in the high TFA diets and the rTFA provided 1.5% of daily energy intake in the moderate rTFA diet. Finally, the control diet provided 0.8% of daily energy intake from rTFA and 0% from iTFA. Results showed that plasmaLDL-C concentrations were significantly higher after the high- rTFA diet than after the control (P<0.03) or the moderate- rTFA diet (P<0.002). Plasma LDL-C concentrations were significantly higher (P<0.02) after the iTFA diet than after the moderate-rTFA diet. PlasmaHDL-C concentrations were significantly lower (P<0.02) after the high rTFA diet than after the moderate-rTFA diet. All risk factors were comparable between the control and the moderate-rTFA diets.

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Author, Year, Study Design, Class, Rating	Study Population and Location	Intervention	Significant Results	Limitations
Chardigny J, Destaillats F et al, 2008 Study Design: Randomized, double-blind, controlled, cross-over trial Class: A Rating: 	N=40 normolipidemic French subjects (21 women, 19 men). Mean age: 27.6±7.1 years. Attrition: 9%. Location: France.	**rTFA vs. iTFA** One week run-in period; two, three-week experimental periods; one-week wash-out period. rTFA and iTFA fed (11-12g per day, ~5% of daily energy) daily in the form of 20g butter,100g cheese and 22g cookies. Compliance assessed via questionnaire and plasma assay for TFA in cholesteryl-esters.	Compared with iTFAs, rTFAs ↑ HDL-C (P<0.012) and LDL-C(P<0.001) in women, but not in men.	Limited generalizability. Limited to young and healthy people with good lipid profile only and no good controls were used.
Jakobsen M, Bysted A et al, 2006 Study Design: Narrative Review Class: R Rating: 	Not applicable.	**Reviewed findings:** • Three prospective cohort studies • One case control • One descriptive study. Studies examined the effects of different quintiles of intake of rTFA and iTFA on CHD risk factors.	Two prospective cohort studies found: Inverse association between energy-adjusted rTFA intake and risk of CHD: Willett WC et al, 1993 reported RR of CHD for the highest vs. the lowest quintile of energy adjusted rTFA to be 0.59 (95% CI 0.30-1.17). Pietinen P et al, 1997 reported that the RR of coronary death for the highest vs. the lowest quintile of energy adjusted rTFA was 0.83 (95% CI 0.62-1.11). Ascherio A et al, 1994 (case-control study) reported that the RR of MI for the higher vs. lowest quintile of energy-adjusted rTFA intake was 1.02 (95% CI 0.43-2.41). Findings imply that intake of rTFA, as C18:1,t11 (vaccenic acid) is innocuous or even protective against CHD. Oomen CM et al, 2001 (prospective cohort) found NS direct associations between intake of rTFA and iTFA and risk of CHD; i.e., for 0.5% higher level of energy intake from rTFA, RR of CHD was 1.17 (95% CI 0.69-1.98) and, for iTFA, RR was 1.05 (95% CI 0.94-1.17).	Not a systematic review. Article selection methods not described.

Motard-Belanger A, Charest A et al, 2008 Study Design: Double-blind, randomized, crossover controlled trial Class: A Rating: 	N=38 normolipidemic Canadian men. Mean age: 32.8±15.0 years. Attrition: 20%.	rTFA vs. iTFA: High and Moderate concentrations. Four isocaloric experimental diets: High in rTFA (10.2g per 2,500kcal) Moderate rTFA (4.2g per 2,500kcal) High in iTFA (10.2g per 2,500kcal) Low in iTFA (2.2g per 2,500kcal) (control diet). All meals were provided to participants. Based upon checklist provided, 99.9% of food provided was consumed.	High-rTFA: Plasma LDL-C significantly higher after the high- rTFA diet than after the control (P<0.03) or the moderate- rTFA (P<0.002) diet. Plasma LDL-C concentrations significantly higher (P<0.02) after the iTFA diet than after the moderate-rTFA diet. Plasma HDL-C significantly lower (P<0.02) after the high rTFA diet than after the moderate-rTFA diet.	Funded in part by the dairy industry. No mention of intent to treat statistics analysis. Study involved only healthy males. Small sample size.

Research Design and Implementation Rating Summary
For a summary of the Research Design and Implementation Rating results, click here.

Worksheets

Chardigny JM, Destaillats F, Malpuech-Brugère C, Moulin J, Bauman DE, Lock AL, Barbano DM, Mensink RP, Bezelgues JB, Chaumont P, Combe N, Cristiani I, Joffre F, German JB, Dionisi F, Boirie Y, Sébédio JL. Do Trans fatty acids from industrially produced sources and from natural sources have the same effect on cardiovascular disease risk factors in healthy subjects? Results of the Trans Fatty Acids Collaboration (TRANSFACT) study. *Am J Clin Nutr.* 2008 Mar; 87 (3): 558-566.

Jakobsen MU, Bysted A, Andersen NL, Heitmann BL, Hartkopp HB, Leth T, Overvad K, Dyerberg J. Intake of ruminant trans fatty acids and risk of coronary heart disease-an overview. *Atheroscler Suppl.* 2006 May; 7 (2): 9-11. Epub 2006 May 18. Review.

Motard-Belanger A, Charest A, Grenier G, et al. Study of the effect of trans fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease. *Am J Clin Nutr. Mar* 2008; 87 (3): 593-599.